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EXERCISE HYPERVENTILATION AND CENTRAL SLEEP APNEA IN HEART FAILURE PATIENTS: RELATIONSHIP TO NEUROHUMORAL ACTIVATION

ACC Poster Contributions
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Background: Exercise hyperventilation is frequent in heart failure (HF) patients. Central sleep apnea (CSA) is also frequent in HF and characterized by hyperventilation followed by compensatory apnea. Serum concentrations of norepinephrine (NE) and endothelin (ET) are elevated in HF patients but their relationship to ventilation is not well established. We evaluated the interactions between circulating NE and ET with hyperventilation and CSA in HF patients.

Methods: Consecutive ambulatory HF patients (n=30) were enrolled to undergo polysomnography for detection of CSA, cardiopulmonary exercise testing for measurement of peak oxygen consumption (VO2), peak end-tidal carbon dioxide concentration (PetCO2) and minute ventilation-carbon dioxide production relationship (VE/VCO2 slope), and measurement of NE and ET-1 concentration. CSA was defined as an apnea-hypopnea index >15 events/hour. Subjects with obstructive sleep apnea were excluded.

Results: Compared to subjects without CSA (n=17), subjects with CSA (n=13) tended to be male (77 vs 59%, p=0.44), were slightly older (65.5 vs 57.7 years, p=0.08), had lower left ventricular ejection fraction (24.6 vs 29.7%, p=0.09), higher BNP concentration (468 vs 198 pg/ml, p=0.03) and similar peak VO2 consumption (19.7 vs 17.4 ml/kg/min, p=0.36). Subjects with CSA had a lower PetCO2 (31.9 vs 38.6 mmHg, p=0.03), higher VE/VCO2 slope (38.7 vs 30.3, p=0.04), higher ET-1 concentration (2.2 vs 1.1 pg/ml, p=0.02) and a trend toward higher NE concentration (432 vs 321 pg/ml, p=0.15). Both NE and ET-1 concentration correlated with PetCO2 (r^2=0.47, p<0.01 and r^2=0.51, p<0.01) and VE/VCO2 (r^2=0.52, p<0.01 and r^2=0.59, p<0.01), and this correlation persisted after controlling for peak VO2 consumption.

Conclusions: These data suggest that CSA is associated with daytime hyperventilation, which correlates well with both NE and ET concentration. The role of neurohumoral activation in modulation of breathing in HF requires further clarification.